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REVIEW

Alpha-lipoic acid effect on leptin and adiponectin concentrations: a systematic review and meta-analysis of randomized controlled trials

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Abstract

Background New evidence suggests that dysregulation of adipocytokines caused by excess adiposity plays an important role in the pathogenesis of various obesity comorbidities. Our aim in this meta-analysis was to determine the effect of alpha-lipoic acid (ALA) supplementation on serum levels of leptin and adiponectin.

Methods We searched Scopus, PubMed, Google Scholar, and ISI Web of Science from inception up to July 2019. Mean difference for leptin and adiponectin were calculated by subtracting the change from baseline in each study group. Summary estimates for the overall effect of ALA on serum leptin and adiponectin concentrations were calculated using random effects model. Results were presented as weighted mean difference (WMD) and their 95% confidence intervals (CI). Between-study heterogeneity was examined using the I^2 statistics.

Result Eight studies were included in systematic review and seven studies in meta-analysis. The overall effect suggested a significant decrement in serum leptin concentrations (WMD = − 3.63; 95% CI, − 5.63, − 1.64 μg/ml; I^2 = 80.7%) and a significant increase in serum levels of adiponectin (WMD = 1.98 μg/ml; 95% CI, 0.92, 3.04; I^2 = 95.7%). Subgroup analyses based on age showed a significant reduction in leptin levels only in younger adults, and subgroup analysis based on duration indicated in studies with a duration of more than 8 weeks adiponectin levels increased significantly and leptin levels decreased significantly.

Conclusion Our results revealed ALA decreased leptin and increased adiponectin especially in studies lasted more than 8 weeks. We still need more studies with different ALA dose, intervention duration, and separately on male and female.

Keywords Alpha-lipoic acid · Leptin · Adiponectin · Adipokines

Introduction

Body fat tissue is considered an endocrine organ which secretes various bioactive substances known as adipocytokines or adipokines such as leptin, adiponectin, and tumor necrosis factor-a (TNF-a). There is strong evidence suggesting that dysregulation of adipocytokines caused by excess adiposity plays an important role in the pathogenesis of various obesity comorbidities such as diabetes, metabolic syndrome, coronary vascular disease, and non-alcoholic fatty liver [1–3].

Serum concentrations of all adipokines, except for adiponectin, are increased during obesity and obesity-related complications. Based on the antiatherogenic, anti-inflammatory, and insulin-sensitizing properties of adiponectin, its increment may ameliorate obesity complications [4]. Unlike adiponectin, leptin has proinflammatory properties and induces insulin resistance and inflammation in metabolic syndrome [5] and may cause the progression of hepatic steatosis and fibrogenesis [6]. Leptin is mainly secreted by adipocytes, and its concentration is proportionally to the mass of adipose tissue [7]. Other tissues such as placenta, ovaries, mammary epithelial cells, skeletal muscles, stomach, pituitary glands, and liver also secret leptin [8].

Pharmacological intervention in combination with physical activity and lifestyle modification such as calorie restriction and dietary modifications may influence plasma concentrations of adipokines [9–11].

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